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IDIOPATHIC COR PULMONALE IN AFRICAN CHILDREN

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Cor pulmonale in children is comparatively rare and there are not many conditions that enter into the differential diagnosis. The most important causes are chronic lung disease (unresolved pneumonia, emphysema, etc.), and pulmonary hypertension, which may be primary or secondary to congenital heart disease. In addition, in recent years reports have appeared of the Hamman-Rich syndrome in infants and children.[1-5]

We report 5 cases of a cardio-respiratory syndrome in children whose clinico-pathological features did not fall into the usual categories of cor pulmonale.

CASE 1

V.M., an African male aged 25 months, weighing 26 lb., was admitted on 9 September 1958 to Baragwanath Hospital with a history of cough and swelling of the whole body of 2 days' duration. He had been admitted to another hospital for the same complaint on 2 occasions in the previous 6 months. He was extremely dyspnoeic, cyanosed, and oedematous with a raised jugular venous pressure. There was gross cardiomegaly, hepatomegaly and ascites, with coarse crepitations in both lung bases. The cardiac impulse was strongest over the lower end of the sternum, and there was a grade I apical systolic murmur. The peripheral pulses were good and the extremities warm. No finger clubbing was present. The heart rate was 114 with regular rhythm and the blood pressure was 80/40 mm.Hg. The

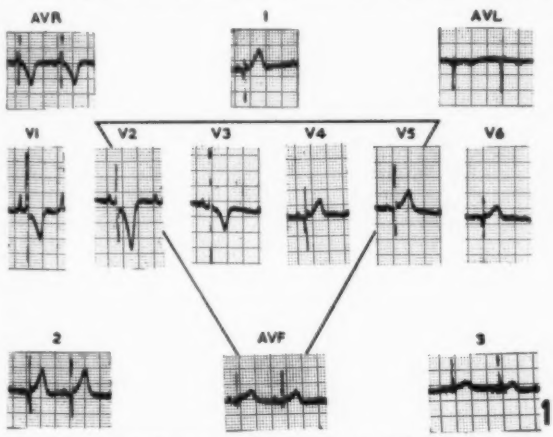

Fig. 1. Electrocardiogram showing right axis deviation — + 120°; right atrial enlargement — tall peaked P waves V_1 - V_2; marked right ventricular hypertrophy and strain — systolic overload (tall R waves with depressed ST segments and inverted T waves V_1 - V_4).

ECG pattern showed a large right ventricle (Fig. 1) which was confirmed radiologically (Fig. 2). He was given oxygen and digitalis and after 48 hours had responded very well, with disappearance of the cyanosis and the systolic murmur. The 2nd pulmonary sound was accentuated, but unsplit. At this time a loud early diastolic murmur was audible in the 2nd left interspace, but disappeared after 24 hours. During convalescence an attempt at cardiac catheterization was made, but had to be abandoned because the child developed a cardiac arrhythmia. Over the next 2 or 3 weeks the systolic murmur over the praecordium and the diastolic murmur in the 2nd

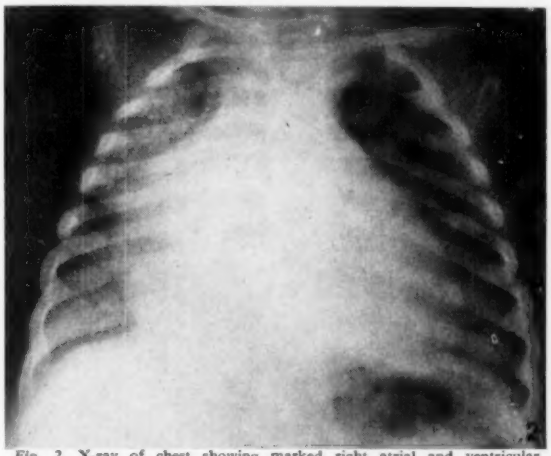

Fig. 2. X-ray of chest showing marked right atrial and ventricular enlargement.

left interspace came and went without explanation. He recovered completely from the congestive cardiac failure and left hospital 32 days after admission.

He was re-admitted 6 weeks later with signs of cor pulmonale and with bronchopneumonia. He responded fairly well to treatment and was sent home on the 23rd hospital day. A month after leaving hospital he was again re-admitted with severe cor pulmonale which failed to respond to treatment, and died on the 28th hospital day.

Autopsy Findings

There was slight oedema of the lower limbs. The heart was markedly enlarged (weight 225 G., expected weight about 56 G.) from hypertrophy and dilatation of the right ventricle and right auricle. The anterior wall of the right ventricle measured 9 mm. at the outflow tract. The epicardium, myocardium, and all the valves appeared normal, but the main pulmonary trunk was slightly wider than the aorta. There was no evidence of any congenital cardiac defect.

The lungs (combined weight 310 G.) were almost double the expected weight. The pleural surfaces were smooth and presented a mottled appearance, dark-red areas alternating with paler greyish areas. Scattered sub-pleural petechiae were present. There was slightly increased resistance on cutting the lungs, and the parenchyma also presented a mottled-red appearance. In addition there were about 12 small, round, greyish-white nodules, measuring about 1 mm. in diameter, scattered throughout the parenchyma of both lungs.

The hilar and tracheobronchial lymph nodes were of normal size and appeared healthy. The pulmonary vessels were normal and the remaining organs showed congestion only.

Microscopic Examination

The heart showed hypertrophy of the muscle fibres of the right ventricle. There was no evidence of rheumatic carditis and the valves were normal.

The lung picture was fairly uniform in multiple sections from both lungs. There were numerous macrophages in most of the alveoli. They were large, many had lobed nuclei, and many were multinucleated (Figs. 3 and 4). Their cytoplasm contained granules which were periodic-acid-Schiff (PAS) positive, and remained so after diastase digestion. A few macrophages showed cytoplasmic vacuoles; some contained small quantities of lipid and many contained haemosiderin granules. No inclusion bodies were found. The alveolar septa were thickened, and the septal capillaries congested and increased in number. There was minimal septal cellular infiltration, consisting of occasional lymphocytes, histiocytes and rare neutrophil leucocytes (Fig. 5). Some alveoli showed swelling of their lining cells. Silver impregnation showed a moderate increase in the thickness and number of reticulin fibres. In some areas these formed a broad interlacing meshwork surrounding the septal capillaries (Fig. 6).

Sections of the small nodules showed them to be granulomata with necrotic eosinophilic centres. Outlines of the septal capillaries were still visible in the granular debris. Surrounding

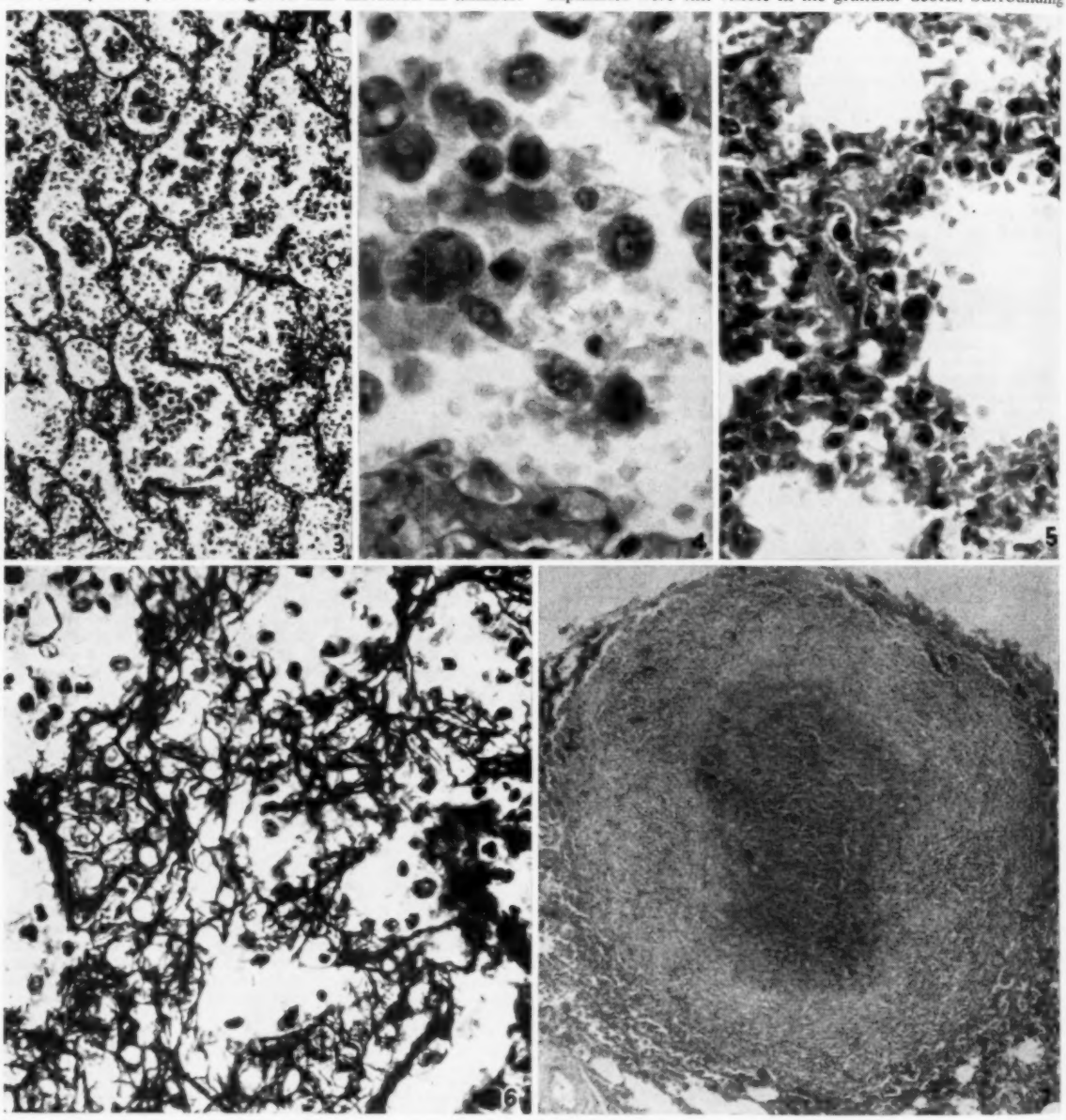

Fig. 3. Low-power section of lung showing numerous macrophages in the alveoli (haematoxylin and eosin × 120).
Fig. 4. High-power photograph of Fig. 3 showing numerous large macrophages in the alveoli.
Fig. 5. Thickened alveolar septa with moderate cellular infiltrate (haematoxylin and eosin × 120).
Fig. 6. Moderate reticulin proliferation in alveolar septa (Gordon and Sweet silver impregnation × 480).
Fig. 7. Section of a granuloma showing necrotic centre (haematoxylin and eosin × 45).

the necrotic centres there was a zone of macrophages, fibroblasts and occasional foreign-body-type giant cells. Stains for tubercle bacilli and fungi were negative (Fig. 7).

The bronchi and bronchioles were normal. The pulmonary arteries were in the main normal, although a few small branches showed slight recent intimal thickening, and, in 1 or 2 instances, recent antemortem thrombi.

The hilar lymph nodes showed non-specific reactive hyperplasia with marked congestion and some haemosiderosis.

Sections of the remaining viscera showed passive congestion only.

SUMMARY OF CLINICAL AND PATHOLOGICAL FINDINGS IN REMAINING 4 CASES

The clinico-pathological features of these 4 cases were those of cor pulmonale of unknown aetiology. The ages of the children varied from 10 months to 2½ years, and they all presented at hospital with cough, oedema and dyspnoea, all of about 2 weeks' duration. All the subjects presented during a 2-month period.

Clinically, the signs were those of cor pulmonale with congestive cardiac failure. While in the ward, over a period of a few months, the cardiomegaly increased rapidly. Laboratory investigations were negative. There was no evidence of whooping cough, bronchiectasis, emphysema, asthma, fibrocystic disease, rheumatic fever or tuberculosis. Three patients gave a past history of bronchopneumonia and 1 had had whooping cough 1 year previously. The family histories were non-contributory. These children failed to respond to the usual measures adopted for cardiac failure, and they all died 1½ - 11 months after the onset of symptoms.

At autopsy, the pertinent findings were confined to the heart and lungs. The hearts were grossly enlarged owing to right ventricular hypertrophy, the weights varying from 1½ to 4 times the expected weight. There were no congenital cardiac abnormalities. The lungs were uniformly enlarged, heavier than normal, and showed increased resistance to cutting. The parenchyma showed a diffuse mottling, dark-red areas alternating with those of normal colour. Some of the other cases showed focal bronchopneumonia. The hilar lymph nodes were normal in all cases. The remaining organs showed the effects of chronic passive congestion.

Microscopic examination of the hearts showed muscle hypertrophy only. There was no evidence of a rheumatic carditis. Sections of the lungs showed a fairly uniform picture. Numerous macrophages were present in the alveoli and these often contained haemosiderin and some PAS-positive material. Occasional macrophages contained small amounts of fat. Areas of intra-alveolar haemorrhage were frequent, and scanty hyaline membrane was seen in one case. The alveolar septa showed proliferation of the capillaries. None of these cases showed the granulomata observed in case 1. There was no evidence of tuberculosis, and the features were not those of alveolar proteinosis. Stains for fungi, bacilli and inclusion bodies were uniformly negative.

Bronchopneumonia of varying severity was seen in most cases. There was no evidence of pneumocystis carinii. Pulmonary vascular changes, when present, were mild and probably secondary to pulmonary hypertension.

DISCUSSION

The difficulty in these cases was that of identifying the disease process in the lungs. There was perhaps some resemblance to diffuse interstitial fibrosis of the lungs of the Hamman-Rich type.[1] Although rare in children, several cases have been described under the age of 10 years, the youngest being 7 months,[2-5] and recently a similar condition has been described in premature infants.[6] Heppleston[7] was the first to note that the fibrous proliferation in the alveolar septa in the Hamman-Rich syndrome was of reticular rather than collagenous type. Golden and Bronk[8] considered that the fundamental lesion was reticular hyperplasia associated with vascular changes. True fibrosis does occur, but tends to be focal rather than diffuse and does not obscure the fundamental vascular and reticular hyperplasia.

It is possible that some of the changes in the alveolar septa in our cases could have been brought about by oxygen administration. Pratt[9] presented evidence to show that, with oxygen inhalation for as little as 2 days, pulmonary alterations, consisting of capillary congestion and proliferation, may be observed. After continuous inhalation for approximately 2 weeks, diffuse fibrosis has been encountered. The lesions are thought to be reversible until fibrosis supervenes. Our subjects were admitted with cor pulmonale and then given oxygen. While the oxygen could thus not have caused the initial heart failure, it may have played a part in accentuating the reticular proliferation in the lungs.

Intra-alveolar haemorrhage was a frequent finding in our cases and macrophages containing haemosiderin were prominent. These changes were, however, probably from the congestive cardiac failure and not severe enough to warrant a diagnosis of idiopathic pulmonary haemosiderosis. The changes in the pulmonary elastic tissue were not remarkable, and foreign-body giant cells containing engulfed elastic tissue were not observed.

The significance of the granulomata in case 1 remains uncertain.

SUMMARY

Five cases of cor pulmonale of obscure aetiology in children are described. A possible relationship to the Hamman-Rich syndrome is discussed.

We wish to thank the Superintendent of Baragwanath Hospital for permission to publish, the Radiology Department for the X-rays, Prof. B. J. P. Becker for advice, and the Director of the South African Institute for Medical Research for facilities granted. Mr. M. Ulrich of the photographic department of the SAIMR produced the photographs.

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TRANSVAAL SOCIETY OF PATHOLOGISTS

SUMMARIES OF SCIENTIFIC PAPERS*

1. PULMONARY ALVEOLAR PROTEINOSIS

REPORT OF A CASE AND A HISTOCHEMICAL INVESTIGATION

PROF. B. J. P. BECKER, *Department of Pathology and Microbiology, University of the Witwatersrand, Johannesburg*

* Read at a meeting of the Society held at Onderstepoort, Pretoria, on 11 March 1961.

1. The first case to occur in South Africa of a newly described disease, pulmonary alveolar proteinosis, was described.

2. Histochemical investigation shows that the alveolar contents contain abundant mixed lipids lying in a large pool of mixed mucins and traces of protein.

3. The histological appearances include a previously undescribed elastolysis and this, together with marked alveolar-cell

proliferation and degeneration, could account for the unique alveolar contents.

4. The clinical history and histological appearances suggest a persistent subacute or chronic virus infection.

2. ECHINOCOCCUS IN SOUTH AFRICA

MISS A. VERSTER, *Department of Parasitology, Onderstepoort Research Institute*

1. The incidence of hydatidosis in livestock was discussed and found to vary from centre to centre.

2. The species identification of these parasites in South Africa is still doubtful. Hydatid cysts of bovine origin fed to various carnivorous hosts resulted in the appearance of parasites which morphologically resemble *Echinococcus granulosus* (Rud. 1801) Batsch, 1786, whereas hydatid cysts of ovine origin resulted in the apperance of parasites resembling *E. lycaontis*, Ortlepp, 1934.

3. REGRESSION OF THE CORPUS LUTEUM AS A CAUSE OF ABORTION IN ANGORA GOATS IN SOUTH AFRICA

DR. S. W. J. VAN RENSBURG, *Stock Diseases Research Fund, Onderstepoort*

The increasing incidence of abortions in Angora goats, which is as high as 70% in some flocks, is becoming a serious menace to the mohair industry in this country.

Intensive research in the past 5 years has eliminated infection; environmental conditions such as shearing, dosing, handling, dipping, and climate; and nutritional defects, as causal agents. The evidence suggests an inherent weakness or predisposition on the part of the affected ewes.

Postmortem examination revealed marked regression and even complete absence of the corpus luteum in aborting ewes. This was confirmed by performing laparotomies on ewes at various stages after service and conception, and subsequent to abortion and normal parturition.

Complete luteal regression and atresia can be found as early as 60 days after service and conception. In aborters, the corpus luteum, if present, is small, pale and firm, while in normal kidding ewes it maintains its size, yellow colour, vascularity and soft consistency up to 2 weeks postpartum.

Affected ewes usually become habitual aborters, losing the foetus in successive seasons; some may actually conceive and abort up to 3 times in the same season.

A significant feature is the marked development of Graafian follicles in the ovaries of such ewes. This is often accompanied by oestrus at the time of abortion.

K. M. van Heerden[1] found marked histological aberrations in the corpora lutea and hypophyses of aborters. The stroma of the corpus luteum is very prominent, and the lutein cells show pyknotic nuclei and some karyorrhexis, while the cytoplasm is homogeneous, with strong eosinophilic colouration, indicating regression and even complete atresia of the corpus luteum.

In the hypophysis of the aborters the acidophiles are small and not densely packed, while the cytoplasm is homogeneous and orange-coloured with pyknotic nuclei, thus showing marked depletion of acidophilic activity. On the other hand the basophiles reveal marked activity, being more numerous, especially centrally, with large cells.

The nucleus becomes pyknotic, while the cell is still large and the cytoplasm vacuolated.

The essentiality of the corpus luteum throughout pregnancy in the goat has been established. It is known that luteotrophic hormone, which is responsible for the maintenance of the corpus luteum and the secretion of progesterone, is secreted by the acidophiles, and follicle-stimulating hormone by the basophiles.

It is concluded that abortion in Angora goats in South Africa is due to premature regression and atresia of the corpus luteum graviditatis. The available evidence suggests that this has a genetic basis, and that inbreeding plays an important rôle.

1. van Heerden, K. M. (1961): 'Investigations into the cause of abortions in the Angora goat in South Africa.' Thesis for degree of D.V.Sc.

4. THE FOLIC-ACID CLEARANCE TEST AS AN INDEX OF FOLIC-ACID DEFICIENCY

DRS. K. STEVENS, J. METZ and V. BRANDT, *South African Institute for Medical Research*

5. SOME ASPECTS OF EXPERIMENTAL ASBESTOSIS

DR. J. C. WAGNER, *Pneumoconiosis Research Unit, Council for Scientific and Industrial Research and South African Institute for Medical Research*

The comparison of the development of asbestotic lesions in man and various experimental animals was demonstrated.

6. 'LAMKRUIS'

DR. T. F. ADELAAR, *Department of Toxicology, Onderstepoort Research Institute*

7. TOXOPLASMOSIS IN SOUTH AFRICAN DOGS

DR. J. D. SMIT, *Department of Pathology, Onderstepoort Research Institute*

A brief review of the literature on toxoplasmosis was given. The essential pathological lesions were described. Seven cases of toxoplasmosis in dogs in South Africa were described. The incidence is apparently high in the Pretoria area. The differential diagnosis includes the usual 'distemper complex', distemper, biliary fever, Rhubarth's disease, Rickettsia canis, and histoplasmosis. In the acute cases the respiratory symptoms predominate and in the chronic form a reactive hyperplasia of the lymphoid tissue is the outstanding feature of the disease.

8. SUBACUTE INCLUSION ENCEPHALITIS

DRS. N. S. F. PROCTOR and J. C. E. KAUFMANN, *Department of Neuropathology, South African Institute for Medical Research*

This disease was first described in the USA by Dawson in 1933. Further cases were subsequently recorded in England and North America. Van Bogaert reported cases from the continent of Europe presenting similar clinical and pathological features, which he called subacute sclerosing leuco-encephalitis. It has subsequently been accepted that these cases represent the same disease process.

Seven cases of this disease occurring in the Transvaal and Northern Orange Free State have been investigated by us.

The condition develops essentially in children and adolescents. In our cases the ages ranged from 4 to 14 years. All were males in this group, but females may also be affected. One Bantu and one Coloured child were included in this series, the rest being European.

The disease varied in duration from 3 months to 13 months, and presented initially either as a febrile illness, myoclonic seizures, or mental changes. There is a progressive cerebral deterioration during the course of the illness and myoclonus occurs at some stage in practically all cases. The electroencephalographic studies are said to show characteristic changes in most instances. The cerebrospinal-fluid analysis is usually of little aid in the diagnosis, being normal in a number of cases.

Pathologically the brain and cord are diffusely involved, with widespread demyelination of the white matter accompanied by reactive glial activity. Focal nerve-cell degeneration with isolated glial 'stars' are found in the grey matter. Occasionally, focal inflammatory-cell reaction is evident in the parenchyma, and usually perivascular lymphocytic cuffing is a prominent feature of the process. Necrosis of grey matter is seen in the more acute cases. The intensity of gliosis varies according to the duration of the disease.

The inclusion bodies are numerous in some cases and difficult to find in others. They may be intranuclear or intracytoplasmic in situation in the nerve cells and may also be found in oligodendroglial nuclei.

Virus studies in a number of the cases both here and overseas have yielded uniformly negative results. The possible association of this disease with canine infection is being investigated.

VAN DIE REDAKSIE : EDITORIAL

OORWEGINGS NA AANLEIDING VAN DIE KONGRES

Die rekords van die kongreskantoor toon aan dat 990 lede van die Mediese Vereniging ingeskryf het vir die Kongres wat gedurende die week 24 - 30 September in Kaapstad gehou is. 'n Groot aantal vrouens van lede en ander familiebetrekkinge was ook teenwoordig. Onder die hooggeplaaste oorsese besoekers was daar 'n aantal uitstaande wetenskaplikes van wêreld-formaat. Ook was die Britse en Kanadese Mediese Verenigings amptelik verteenwoordig op die Kongres.

Die Kongresverrigtinge self het 'n program van ongeveer 270 lesings en voordragte ingesluit, benewens 'n uitstekende wetenskaplike uitstalling, beeldradiovertonings, 'n uitstalling van stokperdjies, 'n handelsuitstalling, en 'n groot verskeidenheid van sosiale geleenthede. 'n Groot en omvangryke organisasie wat baie aan tyd en opoffering van die organiseerders geëis het, was nodig om alles glad van stapel te laat loop.

Die vraag wat begryplikewyse ontstaan, is of daar onder die hedendaagse omstandighede nog die noodsaaklikheid bestaan om so 'n kongres te hou. Sommige twyfel daaraan. Hulle meen dat die rigting waarin die mediese wetenskap ontwikkel het van so 'n aard is dat toekomstige kongresse onvermydelik die vorm moet aanneem van die groepskongresse wat deur ‚homogene' nasionale groepe georganiseer word.

Nou is dit waar dat sommige van die nasionale groepe in die Vereniging baie aktief is en reeds al besondere hoogstaande groepskongresse georganiseer het. Dit is ook waar dat baie groepe al besonder veel bygedra het daartoe om die status en vlak van die mediese praktyk in Suid-Afrika te verhoog. Maar, daar bestaan geen twyfel nie dat daar vir die ontwikkeling van die sogenoemde broederskap van geneeshere veel meer nodig is as wat die groepskongresse kan bied.

Ons leef in 'n tyd waarin daar in die medisyne, soos ook op soveel ander gebiede van die lewe, die neiging is om te ontwikkel in die rigting van al meer en meer gespesialiseerde kennis. Nou is hierdie neiging in sigself nie verkeerd of onwenslik nie. Trouens, vermeerderde kennis ontstaan op die basis van grondiger studie. En die omvang van die mediese wetenskap word so groot dat geen enkele persoon dit alles kan omvat nie. Maar, die kernbegrippe wat in hierdie verband nie uit die oog verloor moet word nie is die begrippe van balans en perspektief. Dit is nie net die diepte van 'n mens se kennis wat sy status as mens en geleerde bepaal nie, maar ook die omvang van sy kennis, en bowe alles sy menslikheid.

Omdat die mediese praktyk nog in so 'n groot mate 'n lewenskuns is, eerder as 'n eksakte wetenskap, moet daar dus geleenthede wees in ons mediese professionele omgang om ook 'n insig te kry in die breër behoeftes van al ons kollegas.

'n Algemene mediese kongres maak sulke geleenthede moontlik op 'n akademiese sowel as op 'n maatskaplike vlak. En daarin lê sy groot waarde. In die seksionele vergaderings is daar 'n kans vir deskundiges op verskillende gebiede om hul kennis te deel met mededeskundiges. In die voltallige sittings is daar geleentheid om die soort probleme te bespreek wat van belang is vir alle geneeshere in alle vertakkinge van die medisyne.

Daar is egter iets anders wat op 'n veel subtieler vlak plaasvind. Oor 'n koppie tee of 'n bord kos word gedagtes gewissel en gesprekke gevoer wat lei tot die vorming van nuwe kennisse en vriendskappe en tot die ontstaan van insigte wat anders nie moontlik sou wees nie. Die tradisie van kongresse van hierdie aard het juis ontstaan omdat die wêreld 'n behoefte gevoel het aan beskawingsinvloede van hierdie aard.

Dat daar gebillikte punte van kritiek teen die hou van so 'n algemene kongres geopper kan word, is 'n feit wat natuurlik nie weggeredeneer kan word nie. Dit is, byvoorbeeld, waar dat daar die neiging is om te veel voordragte toe te laat en om te min tyd beskikbaar te stel vir indiwiduele voordragte. Kritiek van hierdie aard kan vermenigvuldig word.

Maar, as ons alles teen mekaar opweeg, moet ons erken dat ons nie graag die verdwyning van ons kongresse wil sien nie. Miskien moet ons juis nou ons kritiese gedagtes formuleer sodat ons op 'n konstruktiewe manier steeds verder kan vorder op die pad van vooruitgang; ook dat ons gevrywaar kan word teen Voltaire se uitspraak dat hulle wat nie uit die geskiedenis wil leer nie, gedoem is om hul foute te herhaal. Laat ons dus krities wees, maar laat ons daarby positief en opbouend wees. Want slegs dan sal ons daarin slaag om ons algemene mediese kongresse om te skep in akademiese en kulturele geleenthede waarop 'n ou, geleerde, en tradisieryke professie trots kan wees.

THE DOCTOR AND THE PATIENT—AN ETHICAL APPROACH

A satisfactory doctor-patient relationship is one of the basic necessities of any private practice, or, indeed, of the practice of medicine in any form. We, as doctors, pride ourselves on trying to maintain that relationship at the highest ethical and moral level. Basic tenets of our calling include the Hippocratic Oath, the Ethical Code of the World Medical Association, and other local codes, which act as safeguards for our conduct towards our patients and our colleagues.

In recent years, however, the doctor-patient relationship has been disrupted to some extent by the growth of prepaid medical care. The fact that a third party, be it a benefit society, a medical aid society, or an insurance company, pays all or part of our fees for the services we render to our patients, has meant that the old standards of our profession regarding our monetary dealings with our patients have had to be modified to some degree. A high

percentage of most practices today consist of patients who are helped by prepaid medical care.

This has brought important problems in its train. A number of such patients are continually looking for ways and means of entering into dishonest arrangements with their doctors to defraud the organizations paying their medical fees. Somehow they do not understand that this is highly illegal. They look on these practices almost as a right, and bring strong pressure to bear on their doctors to accede to their requests, so that they can get more from these societies than is their due.

For instance, it is no unusual matter for patients, only recently in benefit, to ask doctors to change the dates of treatment so that an account can fall inside the period during which they are entitled to claim benefits. In some cases, where a contract states that patients must pay for the first two visits for any illness (or for the first few rands of the cost), they ask the doctor to include the visits for several illnesses under one diagnosis, so that they will not have to bear the costs themselves.

A medical certificate is an important document. A doctor's statement on such a certificate is accepted at its face value, and in most instances this is as it should be. Unfortunately, patients frequently ask doctors to give them false certificates. They may have been away from work for some private reason, yet they see no harm in asking for a certificate which will state that they were ill during that period. They may have been genuinely ill, but not for as long as they have been away from work; here again they ask the doctor to extend the period for which he can make out a true certificate. Some patients who are allowed a certain amount of annual sick leave, do not seem to appreciate that this leave is granted only if they are sick. Towards the end of the year in question, they ingenuously ask their doctor to certify them sick so that they can claim the benefit.

Some medical aid societies and insurance companies have a number of exclusions from benefits, for which they will not pay doctors' fees. Prophylactic treatment, for instance, is often excluded. Patients who have had this form of treatment often ask doctors to fill out a claim form with a diagnosis for some illness accepted by the society, so that they will not be responsible for the fees.

Doctors themselves will be able to quote numerous other instances where they have been asked to connive in unethical practices. We are pleased to be able to say that the majority of our colleagues will have nothing to do with such requests, and when they explain matters to their patients, most of them understand that what they propose is morally and legally wrong.

However, there are some doctors who succumb to the temptation from a variety of motives, which, while they are understandable, are none the less reprehensible. Young doctors building up their practices may be afraid to lose patients by not acceding to their demands, while older practitioners may not want to see the results of years of hard work diminishing if their patients leave them. Shortsightedly, they create conditions under which unscrupulous patients are able to blackmail other practitioners with the threat that there is always a doctor who will do what they ask. Thus a vicious circle is created, and the whole profession is brought into disrepute.

Recently, in a court case in the Transvaal, a doctor was fined after he pleaded guilty to 20 counts of fraud involving prescriptions for Railway Sick Fund members. The doctor in question did not profit personally, but this did not alter the fact that he had to face court proceedings. In the course of the case, the judge stated that similar fraudulent practices were in full swing in this doctor's area and elsewhere.

This is an alarming indictment of our profession, and it is high time that we see to it that patients who want to make use of us for their own selfish ends meet with short shrift from *all* doctors. In this way we can continue to hold our heads high and walk fearlessly among all men, upholding the honour of what has been called, not without reason, 'the noble profession'.

STUDIES IN RICKETS IN THE CAPE PENINSULA

II. AETIOLOGY

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Before 1920 the aetiology of rickets was something of an enigma. A few years later, however, an understanding of the beneficial effects of vitamin D,[1,2] and the protective value of ultraviolet rays and sunlight,[3-6] acting through a cutaneous vitamin-D mechanism, did away with much of the confusion. Rickets, however, was later found to be common in tropical and sub-tropical countries where ultraviolet exposure should be adequate.[7-12] Factors which predispose towards rickets might include prematurity, lack of breast feeding, low calcium intake, general undernutrition, hereditary predisposition, and lack of exposure to sunlight.

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PRESENT STUDY

1. *Aims.* We planned to investigate the reasons for the high incidence of rickets in the South African 'Cape Coloured' community, in view of the availability of abundant sunlight. Especially, we wished to examine the relative importance of prematurity (birth weight), breast feeding, intake of calcium, exposure to sunlight, undernutrition and malnutrition, and inheritance (familial incidence, excluding environmental factors as far as possible).

2. *Material.* We have examined 100 children with radiologically proved rickets, referred to us over a period of 2 years, and a control group of hospital outpatients of a

similar age. In some cases the mothers were not capable of providing an adequate history, and these children have been excluded.

3. *Methods.* Most of the data were obtained by direct questioning of the mothers of our patients. Admittedly the data here presented cannot all be accurate, in as much as some mothers were not certain of such factors as the amount of sunlight exposure and the birth weight. However, the discrepancies presumably apply equally to the rickety and control patients.

In assessing calcium intake we have considered only those infants who were not being breast fed and have included only that calcium taken as milk.

Only patients with definite radiological changes were included and, in them, the rickets was confirmed biochemically. Children were aged 3 months - 2 years 10 months, and all suffered from ordinary vitamin-D-lack rickets, to the best of our belief. Renal causes of rickets were excluded as far as possible. Urine was tested as a routine for protein and sugar, although it was not always possible to obtain specimens in outpatients. We were particularly careful to screen all children who (*a*) were over the age of 2 years, (*b*) had a family history of rickets, or (*c*) did not respond to 1 ml. 'ostelin forte' * (= 600,000 units of vitamin D).

During this investigation we did 'discover' 2 siblings in one family with cystinosis, which we shall describe in more detail later, and a family with vitamin-D-resistant rickets.

*Supplied by Messrs. Glaxo-Allenburys (S.A.) (Pty.) Ltd.

The mothers usually remembered fairly accurately how many teaspoons of dried milk they used for preparing feeds, and this was the commonest source of calcium. Two of the dried-milk preparations were analysed and found to contain 276 mg. of calcium per ounce, and, as a rough guide, this was considered to be present in 12 teaspoonsful. An analysis of whole milk has shown it to contain 600 mg. of calcium per pint.

RESULTS

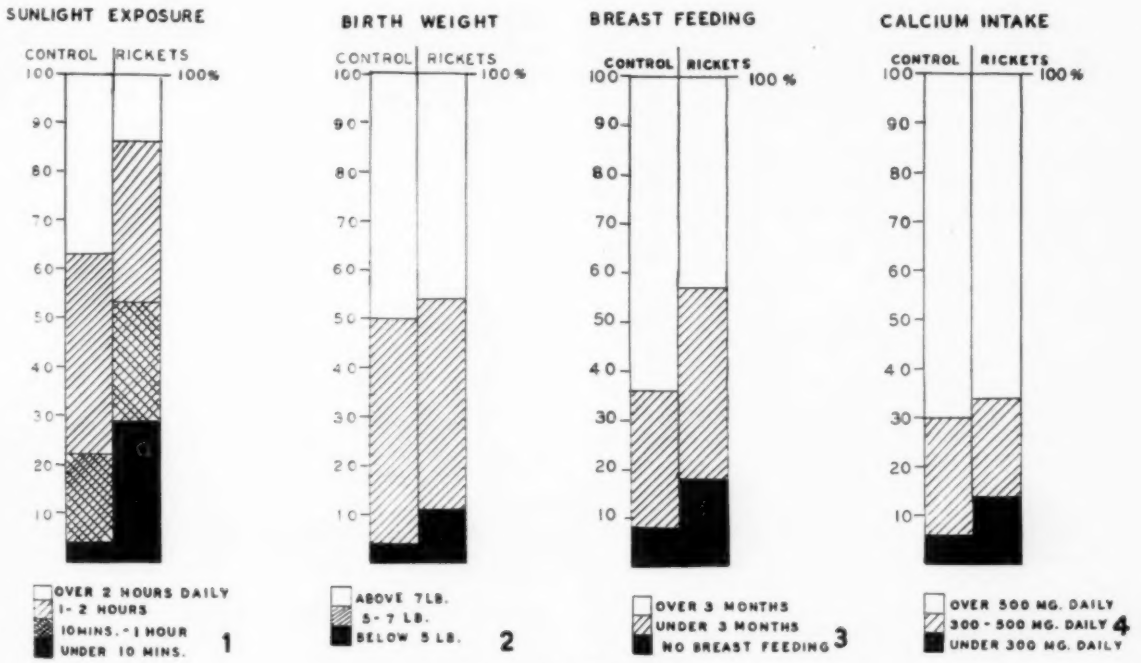
Exposure to Sunlight (Fig. 1)

On the whole, children with rickets saw far less sunlight than controls, more than half being exposed to less than 1 hour daily, and the difference is highly significant ($P = .001$). However, 14% were in the sun for more than 2 hours a day. (There were also 4 control children who were in the sun for less than 10 minutes a day, but had no radiological evidence of rickets.)

Seasonal Variation

Of our total series, 33 were seen in September/October 1959, during an investigation into the significance of craniotabes. These have been excluded from this section, to give an unbiased overall seasonal incidence. Furthermore, only children below 1 year of age are considered here.

Of the remaining 67 children, 51 were found to have rickets during the 5 months, September - January inclusive. These months correspond to spring and early summer in this region.



Figs. 1 - 4. See text.

Most of the children were born in late summer and autumn — 41 out of 53 being born during the 5 months, January - May inclusive.

Birth Weight (Fig. 2)

The children were divided into 3 groups according to their birth weight — below 5 lb., 5 - 7 lb., and over 7 lb. Few children weighed less than 5 lb., but there were 11 in the rachitic group against 4 in the control group. This difference was not significant ($P = .05$). There was apparently no increased liability to rickets in the 5 - 7 lb. group.

Twins

Six of the children in the rachitic group were twins (6%). In 3 of the twin-pairs both children had rickets; in 2 of the twin-pairs only the rickety child had survived, and in the remaining pair the other twin, although brought up in the same way, was normal.

Breast Feeding (Fig. 3)

Many of the rickety children had received no breast feeding at all (17%), and the majority were breast fed for less than 3 months. This is in a community where breast feeding is notoriously prolonged — reflected in the control figures (64% were breast fed longer than 3 months). Comparing the 2 groups, the rickety children were on the breast for a shorter period. The difference between the number of normal children who received breast milk for under 3 months compared to the rickety group is possibly significant ($.01 < P < .02$). Nevertheless there were many children with gross rickets who were still being breast fed.

Calcium Intake (Fig. 4)

Children were again divided into 3 groups — those receiving under 300 mg. per day, 300 - 500 mg. per day, and over 500 mg. per day. There appears to be a greater susceptibility to rickets in those children receiving less than 300 mg. per day, although the numbers in this group are small and the difference between the controls and rickets is barely significant ($P = .05$). Most children with rickets had high intakes of calcium — over 500 mg. daily in two thirds and over 800 mg. daily in one quarter. There was no correlation between the severity of rickets and the calcium intake — some of the children with gross rickets and osteomalacia had intakes of calcium exceeding 1 G. daily.

Combined Factors

There were 37 rickety children in whom more than 1 of these 4 possible aetiological factors were involved, i.e. sunlight exposure of less than 1 hour; breast feeding for less than 3 months; calcium intake under 300 mg. daily, and a birth weight of under 5 lb. In 30 of these, both sunlight exposure and breast feeding were deficient, and in 6 either sunlight exposure or birth weight was a possible factor.

In 25 cases only 1 factor could be incriminated — deficient sunlight in 9, short breast feeding in 13, low calcium intake in 2 and low birth weight in 1.

There were therefore only 4 patients in whom neither defective sunlight exposure nor short breast feeding was a possible aetiological factor, and in whom prematurity or low calcium intake was the sole defect discovered. There were, in addition, 9 children with rickets in whom none of these 4 factors appeared defective.

Familial Rickets

Twenty-three families were investigated for evidence of rickets in more than one sibling. In 13 of these only 1 child was affected, but in 10 families more than 1 sibling had radiological evidence of rickets — in some cases this had healed and only postrachitic deformities remained.

An underlying renal aetiology was discovered in 2 of the families (cystinosis and vitamin-D-resistant rickets). In the remaining 8 families there were no urinary abnormalities, and the rickets healed on small doses of vitamin D. In addition to these 8 instances of what is presumably 'familial ordinary vitamin-D-lack rickets', there were 3 other families in which the mothers stated that at least one other child had deformed legs — in one case she knew her child had been treated in hospital for rickets. These children were not seen by us.

Biochemistry in Mothers (Table I)

The serum-phosphorus and alkaline-phosphatase levels were determined in 17 of the mothers of these children.

TABLE I. BIOCHEMISTRY IN 17 MOTHERS OF RICKETY CHILDREN

Alkaline phosphatase (Shinowara-Bodansky units)	*Phosphorus (mg. per 100 ml.)*	*Alkaline phosphatase (Shinowara-Bodansky units)*	*Phosphorus (mg. per 100 ml.)*
8·4	4·3	2·2	4·9
6·1	4·2	6·0	3·5
16·2	4·5	2·7	5·5
5·1	5·2	5·9	4·3
5·2	4·2	18·3	2·9
2·2	4·1		5·2
3·1	4·2		5·4
13·5	3·9		3·4
6·3	4·4		

All were within the normal range.

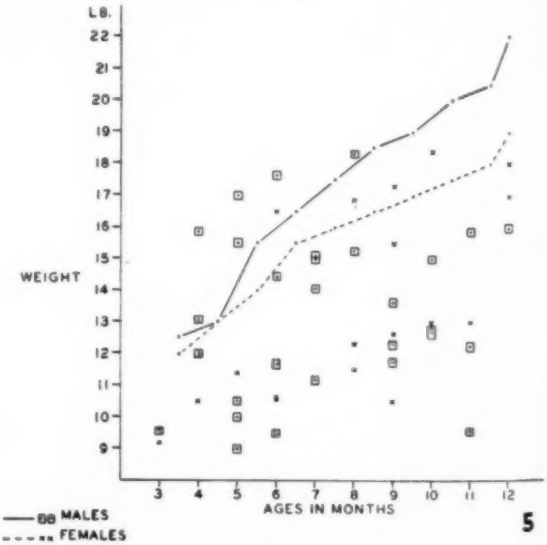


Fig. 5. Weights of children with active rickets compared with mean weights of normal Coloured children. Dotted and solid lines represent, respectively, the mean weights of normal female and male Coloured children under 1 year of age.

Weight of Children with Active Rickets (Fig. 5)

When compared with the mean weights of African and Coloured children (after Salber [15]), nearly all the children with rickets were underweight when first seen, some grossly so.

Serum Proteins (Table II)

The total serum proteins were above 6·0 G. per 100 ml. in 15 out of 21 estimations. Some of these patients had severe rickets with bone rarefaction and fractures. In only 2 children was the total protein below 5 G. per 100 ml., and in only 1 child was the serum albumin below 3·0 G.

TABLE II. SERUM PROTEINS IN G. PER 100 ML. IN 21 ESTIMATIONS

Albumin	*Globulin*	*Total*	*Albumin*	*Globulin*	*Total*
4·5	2·7	7·2	3·1	2·5	5·6
4·2	2·2	6·4	3·8	3·5	7·3*
4·4	1·8	6·2	3·8	2·4	6·2
4·7	3·0	7·7	2·2	1·6	3·8
4·4	1·8	6·2	3·8	2·3	6·1
4·5	2·8	7·3	4·5	2·2	6·7*
3·4	2·0	5·4	4·2	1·4	5·6
4·0	2·2	6·2	3·7	2·5	6·2
3·6	1·3	4·9	4·6	3·0	7·6*
3·2	2·5	5·7	4·5	3·3	7·8*
			4·0	2·0	6·0*

* Electrophoresis carried out. Normal in all 5 patients.

Electrophoretic patterns were done in 5 children who had a history of repeated respiratory or gastro-intestinal infections. The globulin and other fractions were normal in all cases.

DISCUSSION

Sunlight

The seasonal variation in the incidence of local rickets led to lack of sunlight being suspected as an important cause. The healing effect of sunlight has been demonstrated both experimentally in rats,[14] and in humans. Exposure for as little as half-an-hour daily was sufficient to cure rickets in a small group of children investigated by Hess in New York in winter.[4] It is only the short ultraviolet waves which are beneficial,[16] and there are many factors affecting the number of these reaching the earth's surface. Atmospheric absorption is increased by smoke, dust and cloud, and by the distance the rays travel through the atmosphere. The antirachitic quality of sunlight is therefore greatest at midday near the equator, at higher altitudes, and on clear summer days. Irving and Schwartz demonstrated that summer sunshine in Cape Town has marked antirachitic activity; as little as 5 minutes' exposure at 1 p.m. was sufficient to promote healing in rachitic rats.[20] Winter sun, however, had a very low antirachitic activity —exposure of 1 - 2 hours daily failing to heal rickets.

The value of sunlight exposure must also be surveyed in relation to other factors, skin pigment being particularly relevant in this community. Dark skin absorbs less ultraviolet light, and the practical importance of this has been shown in rats. Darkly pigmented rats need longer exposures to sunlight to protect them from rickets when compared with white rats.[17a] It is possible that an urbanized coloured population needs more than a half hour's daily sunlight, especially in winter. Lack of adequate exposure to sunlight appears to be the most definite aetiological factor in these children. Many are carried heavily wrapped on their mothers' backs, where they see little sun. Others are left indoors all day while their mothers work.

Most cases were found in spring and early summer. Any sunlight these children had been exposed to in the previous 6 months was during winter and therefore of poor quality. Likewise, children born in the late summer and autumn are exposed to these poor-quality rays for their first 6 months. Most of the children with rickets in this series were born during that time.

However, half the affected children had been exposed to sunlight for more than 1 hour daily. Even if the minimum requirement to prevent rickets were 2 hours, there are still 14% of children in whom an alternative aetiological factor must be sought.

Milk

Breast milk has always been considered to be superior to cow's milk in preventing rickets. Hess, in 1922, showed that approximately all bottle-fed infants in New York developed rickets in the month of March unless they received specific therapy, whereas 'only' a half of the breast-fed infants showed definite signs at the seasonal peak.[17b] It is not clear why breast milk is superior to cow's milk. The calcium and phosphorus content of breast milk is less than in cow's milk, and it contains very little antirachitic factor.[18] (In premature infants there is evidence that cow's milk has superior antirachitic qualities.[19,20]) In the children we investigated many were breast fed when rickets developed, so that it is certainly not completely protective, although there did appear to be an increased likelihood of rickets, possibly significant, developing in those children who received less than 3 months' breast feeding.

Calcium

The calcium intake was adequate in nearly all the patients. There is no good evidence that lack of calcium ever plays any part in the development of rickets.[21,22]

Prematurity

An increased incidence of rickets among premature infants has frequently been observed.[17] Defective skeletal mineralization has been suggested as a factor (most of the calcium and phosphorus is laid down in the last 2 months of foetal life). Hess showed that the livers of premature infants contain as much antirachitic substance as those of full-term infants. In this series we were unable to demonstrate a significantly increased susceptibility to rickets in premature infants.

Main Aetiological Factors

In comparing these rickety children with controls the most significant aetiological factors appear to be deficient sunlight exposure and breast feeding. Prematurity and deficient calcium intake were not significantly different in the 2 groups.

This conclusion is confirmed by analysing these 4 factors as they affect individual children with rickets. In 62 cases where adequate histories were obtained on all 4 factors, a defect in sunlight exposure and/or breast feeding was present in 58, and in only 4 children was prematurity or deficient calcium intake the sole aetiological agent discovered.

There were, however, 9 additional children in whom all the factors so far considered appeared favourable, so that in these cases we were either misled by an incorrect history, or else there are other important aetiological factors which we have not considered.

Other Factors

The frequency with which more than one member of a family had rickets suggests a possible hereditary factor. The explanation of this may, of course, be merely environmental, in that the children were brought up in the same way with little or no sunlight exposure. We found no evidence of disturbed biochemistry in the mothers' blood (Table I).

During the 2-year period over which this investigation was conducted, only 3 European children were seen with ordinary vitamin-D-lack rickets, possibly indicating a racial susceptibility among both Bantu and Coloured. Difference in upbringing might also explain this.

According to Hess, rickets is rare in the marasmic child.[17c] However, in a recent comprehensive survey of rickets in Japan, the high incidence of rickets in marasmic children was stressed,[22] and most of our children were underweight — some severely. Wayburne and Dean have reported similarly.[24] We believe that the popular conception, that lack of growth protects from rickets, is incorrect.

During various severe illnesses serum levels of calcium and phosphorus may be depressed, returning to normal on recovery. Rickets has also been observed histologically during severe illness.[25] Such rickets may, however, have antedated the illness rather than have been caused by it.

In view of the severity of osteomalacia in some cases, we wondered whether the bone matrix was also involved, especially since the children's protein intake was extremely low in most instances. However, the serum proteins were normal in nearly all the patients examined, including some with the most severe bony rarefaction. The increase in bone density following vitamin-D therapy was often dramatic. We have therefore no evidence to support the suggestion of matrix deficiency.

The electrophoretic pattern of the serum proteins was normal in the cases in which it was evaluated. A deficient γ-globulin fraction did not appear to account for the frequency of superadded infection in rickety children.

SUMMARY

One hundred Coloured and Bantu children with active rickets have been seen over the course of 2 years. When compared to a control group, the most significant difference in possible aetiological factors was the exposure to sunlight, which was far less in the rickety group ($P = .001$). Most of the rickety children were born in the late summer and autumn months. There were more rickety children receiving less than 3 months' breast feeding than controls, the difference being probably significant ($.01 < P < .02$). There was no statistically significant difrerence in birth weight or calcium intake between the 2 groups.

Individual case analysis of rickety children confirmed that deficient sunlight exposure or breast feeding were probably the 2 most important aetiological factors. In only 4 of 62 children in whom adequate histories were obtained were neither of these factors incriminated. There were, however, an additional 9 children in whom all these factors were favourable. Serum proteins were normal in all the children, even in those with gross osseous rarefaction. The mothers of rickety children had normal serum biochemistry. In eight families more than 1 sibling suffered from ordinary vitamin-D-lack rickets.

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LIVER DAMAGE IN FATAL GASTRO-ENTERITIS

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There is little recorded in the literature on liver damage in fatal gastro-enteritis. Various authors have described fatty change,[1] necrosis,[2] hepatitis[3] and even cirrhosis.[4]

In view of the varied opinions expressed, it was felt that a systematic histopathological study of the livers of subjects dying of gastro-enteritis would be of some interest.

MATERIAL AND METHODS

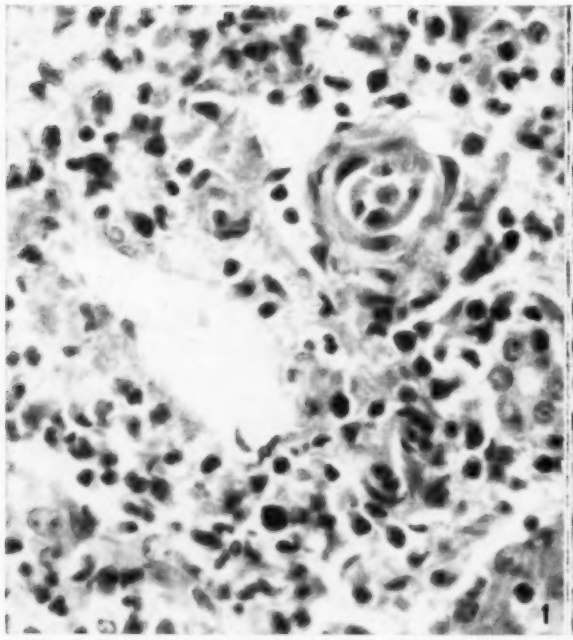
The material examined was obtained from 22 consecutive autopsies on Bantu children dying from the effects of gastro-enteritis. Several blocks of liver were examined histologically, and numerous sections of the common bile duct were also examined. These included a study of the lymphoid tissue and lymphatics in the porta hepatis. In addition an attempt was made to culture pathogenic organisms from postmortem tissue.

RESULTS

The findings are summarized in Table I. The ages of the subjects varied from 2 weeks to 36 months, a period in which the ravages of severe malnutrition, associated with bowel and respiratory infections, result in a high mortality in the Bantu paediatric population. There were 15 females and 7 males. Salmonellae were isolated in 9 cases. *B.proteus* or *E.coli* were cultured in 10 cases, but, since phage-typing was not carried out, the significance of these organisms could not be determined. Sixteen cases showed varying degrees of hepatic steatosis, a common finding in

TABLE I. SUMMARY OF FINDINGS IN GASTRO-ENTERITIS

Case	*Age in months*	*Sex*	*Fatty change*	*Organisms isolated*	*Clinica jaundice*	*Intrahepatic cholestasis*	*Cellular infiltrate in portal tracts*	*Focal necrosis of liver*
1	1	M	Absent	*E. coli*	—	+	—	+
2	2	F	Mild	Salmonella	—	—	—	+
3	10	F	Mild	*B. proteus. E. coli*	—	—	+	—
4	16	F	Marked	Salmonella	—	—	+++	+
5	4	F	Marked	Salmonella	—	+	+	—
6	16	M	Gross	Salmonella	+	+	+	+
7	18	M	Mild	—	—	—	+	—
8	11	M	Moderate	Salmonella	—	—	+	—
9	3	M	Moderate	Salmonella	—	+	+++	+
10	14	F	Gross	*B. proteus. E. coli*	—	—	++	—
11	12	F	Marked	Salmonella	—	—	+	+
12	18	F	Slight	*E. coli*	—	—	++	—
13	3	M	Absent	—	—	—	—	—
14	17	F	Absent	*E. coli. B. proteus*	—	—	+	—
15	8	F	Mild	Salmonella	—	—	+	—
16	24	F	Marked	*B. proteus.* Coliform	—	+	+	—
17	12	F	Absent	Salmonella	—	—	—	—
18	9	F	Marked	Proteus and coliform	—	+	++	—
19	36	F	Gross	—	—	—	+	—
20	½	M	Absent	*B. proteus. E. coli*	—	—	—	—
21	11	F	Moderate	*B. proteus. E. coli*	—	—	+++	—
22	4	F	Moderate	*E. coli*	—	—	+++	—



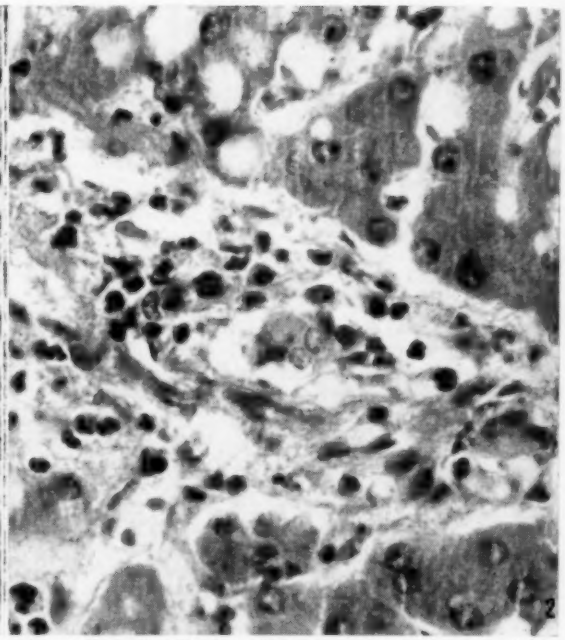


Fig. 1. Section of liver showing heavy cellular infiltrate round portal venule. The bile ducts are normal (haematoxylin and eosin × 600).
Fig. 2. Cellular infiltrate extending from lumen of portal venule through wall into surrounding fibrous tissue (haematoxylin and eosin × 600).

this age group in which kwashiorkor is so prevalent. It was thus impossible to separate the effects of gastro-enteritis from those of malnutrition in the pathogenesis of the fatty liver. A similar situation existed with regard to bile stasis. The 2 cases that showed clinical jaundice also had fatty livers, and of the 6 cases with histological intrahepatic cholestasis, 5 had hepatic steatosis. There is little doubt that the fatty liver by itself can, on occasion, produce bile stasis and clinical jaundice.

Several cases had infiltrates of varying severity in the portal tracts. The infiltrating cells were generally an admixture of lymphocytes and neutrophils, and were present in large numbers in only 4 cases. Mild cellular infiltration of the portal tracts is a frequent autopsy finding in Bantu subjects dying of diseases other than gastro-enteritis, but a heavy infiltrate probably represents the effect of the gastro-enteritis. In such instances the inflammatory cells were spread diffusely in the portal fibrous tissue. It was possible in some cases to see the infiltrate spreading out from the lumen of the portal vein through the wall into the surrounding tissue (Figs. 1 and 2). Involvement of bile ducts was never observed.

Six cases showed focal necrosis, and this was distributed at random in the lobule. In general, the areas of necrosis were small and infiltrated by neutrophils (Fig. 3).

In an attempt to demonstrate an ascending cholangitis, the common bile duct was examined at numerous levels. There was no evidence in any of the subjects that infection reached the liver *via* the biliary tract. The lymph nodes of the porta hepatis showed some degree of cellular infiltration and reactive hyperplasia in 10 cases.

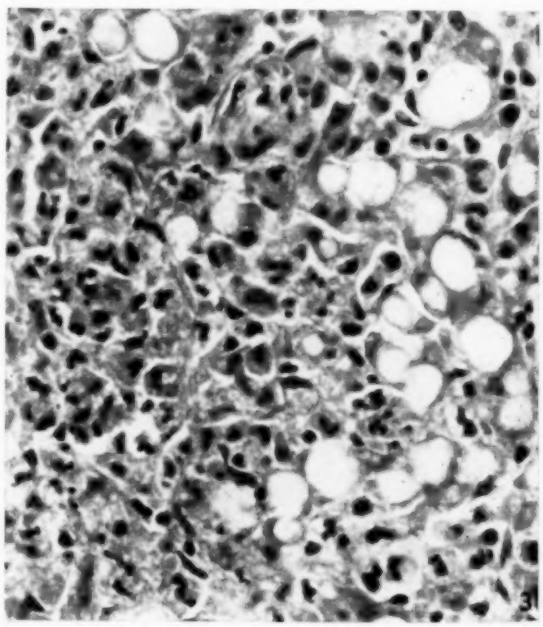


Fig. 3. Area of hepatic-cell necrosis with neutrophil infiltration and fatty change in surrounding liver cells (haematoxylin and eosin × 80).

DISCUSSION

Bonham-Carter,[1] in a study of the liver in fatal cases of diarrhoea and vomiting in infants, described fatty change and fibrosis starting in the portal areas, and foci of miliary necrosis. Since the undernourished infant contracts gastro-enteritis easily, it is possible that the fatty change and portal-tract fibrosis may have been the result of malnutrition, although in this population, where kwashiorkor and fatty liver are commonplace, portal-tract fibrosis and cirrhosis do not appear to be an end-result.[6]

Wainwright[3] described a form of hepatitis associated with jaundice and infantile diarrhoea. The main features were periportal necrosis of parenchymal cells with an infiltration of inflammatory cells and a proliferation of bile canaliculi and fibroblasts. He felt that the hepatitis was caused by a toxin reaching the liver through the blood stream, and suggested a possible relationship between this form of hepatitis and congenital biliary cirrhosis.

Because fatty change in the liver is virtually a constant finding in kwashiorkor, hepatic steatosis could not be used as an indication of liver damage in gastro-enteritis. In fact, the only finding of significance in our cases was the presence of miliary areas of necrosis; these were present in little more than 25% of cases. Mild clinical jaundice or intrahepatic cholestasis is frequently seen in hepatic steatosis, and this could not be shown to result from an ascending cholangitis as has been suggested by Parker.[6] In fact we were never able to demonstrate involvement of the liver *via* the biliary channels, and in cases where the cellular infiltrate in the portal areas was marked, the infection seemed to have reached the liver by way of the portal vein. The infiltrate in the portal tracts was in general not very heavy, but occasionally consisted of large numbers of neutrophils.

CONCLUSIONS

In the majority of cases of fatal gastro-enteritis there are no characteristic findings in the liver. Hepatic steatosis and intrahepatic cholestasis may result from malnutrition and cannot be attributed to the gastro-enteritis. Heavy cellular infiltration in the portal tracts is seen in a proportion of cases, and in such circumstances the infection appears to reach the liver *via* the portal tracts and not the bile ducts. In a small number of subjects areas of miliary necrosis of the liver may be observed.

SUMMARY

The liver lesions in 22 consecutive fatal cases of gastro-enteritis in children are described and the possible pathogenesis discussed.

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INFANTILE CORTICAL HYPEROSTOSIS

S. LEVIN, M.B. (RAND), M.R.C.P. (EDIN.), D.C.H.; and J. FRIEDMAN, M.B. (RAND), D.M.R.D. (LOND.), *Johannesburg*

'O Lord, heal me; for my bones are vexed.'
Psalm 6 : 2.

It is some 15 years since Caffey and Silverman[1] gave the first lucid description of this disorder — so-called because of the age incidence and the characteristic hyperplasia of cortical bone.

Since then considerably more than 100 cases have been reported in the medical literature, though this total is certainly but a small fraction of all cases diagnosed. 'Caffey's disease' is especially well recognized in the USA, where it has also been noted in Negroes and indigenous Indians. The disorder is probably not too rare; in mild or atypical forms it is easily overlooked or misdiagnosed. In this country it has also been recognized — individual radiologists approached have indicated that they have seen rare cases locally, but we have been unable to find any case report in South African literature.

CASE REPORT

In March 1960 an 8-week-old girl was presented for examination because her legs were swollen.

A full-term infant, she appeared well after birth. Nursing failed when the mother and baby returned home on the 10th day, and a milk-water-sugar-cereal mixture was substituted. At 14 days a polyvitamin supplement was added in conventional dosage, and a day or two later the mother observed that the infant's shins were slightly swollen. During the next 6 weeks the shins became markedly convex and tender to the touch, but otherwise she was well, gaining weight, eating satisfactorily, not unduly irritable and not feverish.

Pregnancy was uneventful, but from about the 4th month the father, a representative of a pharmaceutical house, insisted that his wife take daily doses of vitamin capsules ('prenalac'). This she did, more or less faithfully, until term.

The first child, a healthy girl of 2½ years, was born after a normal full-term pregnancy during which vitamin supplements were not taken by the mother.

There was no history of bony disorders in either parent or their relations.

Physical examination of the patient revealed an unhappy 9 lb. infant with astonishingly well-marked sabre tibiae. The subcutaneous tissue overlying the thickened bones was also brawny, but there were none of the usual signs of inflammation. On the dorsum of the right foot there was a solitary *café-au-lait* spot. There was some doubt whether the right ramus of the mandible was swollen. Blood pressure was 90 mm.Hg systolic. There was no fever.

The diagnosis was considered to be either infantile cortical hyperostosis or periosteal neurofibromatosis, and radiology was suggested with these possibilities in view.

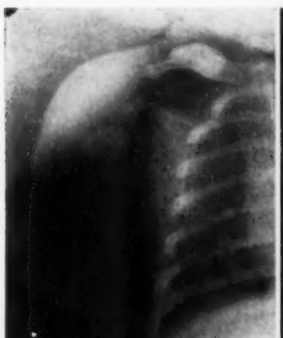
Radiological Findings

Radiographs of the lower limbs demonstrated marked periosteal new-bone formation surrounding both tibiae, affecting the diaphyses and stopping at the level of the metaphyses. The periosteal margins were fairly smooth and no spiculation was observed. The right fibula was similarly affected, but

Fig. 1. A and *B.* Antero-posterior and lateral radiographs of legs, demonstrating the extensive periosteal new-bone formation of both tibial shafts and right fibula with a tendency to onion-peel layering.

the left fibula appeared normal (Fig. 1).

A similar periosteal area of new-bone formation with smooth margins was seen in relation to the middle third of the right humerus (Fig. 2). A slight degree of periosteal reaction was also found on the medial side of the middle third of the left



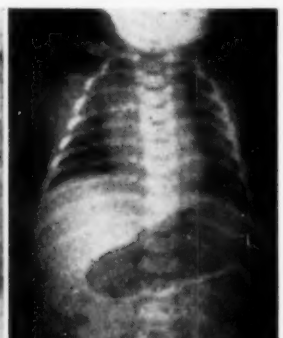


Fig. 2. The periosteal new-bone formation around the right clavicle and humerus is clearly evident.
Fig. 3. The reaction around the right clavicle and the irregular bossing of the mandible, frequently found in infantile cortical hyperostosis, is demonstrated.

humerus. The right clavicle showed marked new-bone formation. The femora, forearms, spinal column and pelvis appeared normal.

The mandible was also seen to be irregular in contour, consistent with patchy zones of periosteal new-bone formation (Fig. 3).

The radiological appearances were typical of an infantile cortical hyperostosis.

Progress

When the diagnosis was established the baby was re-examined. This time there was no doubt that, although the rami of the mandible were not enlarged, small bony knobs projected from the middle of each side. An enlargement of the right clavicle could not be felt clinically; presumably the hyperostosis was limited to the internal surface. No treatment was given.

In early April blood studies revealed the following: Haemoglobin 11·7 G. per 100 ml., WBCs 12,800 per c.mm., neutrophils 32%, lymphocytes 55%, eosinophils 4%, monocytes 7%, basophils 0·5%, platelets numerous, sedimentation rate 25 mm. in the 1st hour, alkaline phosphatase 23·5 King-Armstrong units (normal 10 - 20 units).

A month later the sedimentation rate was 18 mm. in the 1st hour and the alkaline phosphatase was 19 King-Armstrong units.

Over the course of the next few months the tiny knobs on the mandible disappeared, but the patient's shins remained bowed. Teeth erupted at 10 months, and her physical and mental progress was normal. The routine immunizations caused no untoward reactions.

In March 1961 she was re-X-rayed, with findings as follows:

There was complete absence of periosteal new-bone formation. Some residual bowing of both tibiae was present (Fig. 4), but it is confidently expected that this will revert back to

Fig. 4. One year after Fig. 1 was taken. No trace of the periosteal new bone is seen, but some residual bowing is still present. It is confidently expected that, with the passage of time, both tibiae will revert back to normal.

normal. The right clavicle and right humerus appeared perfectly normal (Fig. 5).

DISCUSSION

The disorder occurs equally in both sexes and is usually first manifested at the age of 2 - 3 months, although it has been correctly diagnosed prenatally at 31 weeks' gestation; in that case the foetus was born dead by Caesarean section, and the disease was already well-advanced.[2]

Clearly the aetiology is to be sought within the prenatal period. So far, all studies — bacterial, viral, immunological, nutritional and blood chemical — have been negative.[3] Pathologically the picture is not clear. The bone formation is not secondary to subperiosteal haemorrhage; infective and also benign hyperostotic changes have been suggested.

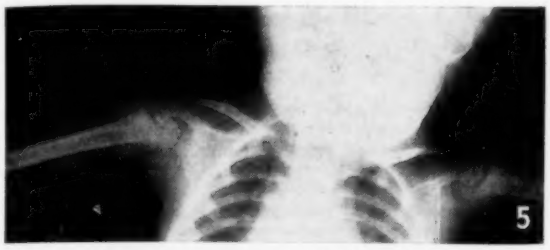


Fig. 5. The normal appearance of the right clavicle and humerus seen one year after Fig. 2 was taken.

Sometimes the picture is virtually indistinguishable from that of osteogenic sarcoma, and there have been instances of the disease affecting a single bone (especially the mandible or scapula) with misleading biopsy reports, resulting in radical surgery with amputation of mandible or limb.[4] In the radiological differentiation it should be noted that, unlike osteogenic sarcoma, there is no spiculation in infantile cortical hyperostosis.

All bones in the body may be subject to the disease, though the spine, carpus, and tarsus appear to be exempt, and the skull is only rarely affected. The mandible is nearly always involved, and the clavicles, tibiae and humeri frequently affected. Also not exempt are the femur, radius, ulna, ribs, fibula and scapula.

In the acute phase of the disease there may be irritability, fever, anorexia and leucocytosis. During resolution the bones may be bowed, but by about 3 years this has nearly always rectified itself. Very rarely, bowing and crippling residua may persist into adulthood and may require orthopaedic correction.

Because the prognosis, though usually benign, may be unpredictable, Caffey[1] suggested that all patients should be treated with cortisone. One death has been reported, apparently from the disease itself, for no other cause could be found at autopsy.[5]

Of late, attention has been directed to the familial incidence of the disease.[6-8] Tampas *et al.*[9] have even noted 11 cases in 2 generations of one family group, though to be sure, half of them had minimal bony involvement and would not have been diagnosed had there been no knowledge of typical instances among relations. Holman and Gerrard[10] observed 13 cases over 3 generations in one family group.

Paradoxically, despite the familial incidence — at least in some cases — the disease appears to be quite new. Sherman and Hellyer[11] reviewed all their X-ray films from 1930 to 1950 and found none that might be relabelled infantile cortical hyperostosis.

The Rôle of Vitamin A

Superficially, infantile cortical hyperostosis is similar to vitamin-A intoxication. Both show hyperostosis, though the distribution differs,[12] and vitamin-A poisoning only appears towards the end of the first year of life, and then only if the preceding months have witnessed the (usually unwitting) intake of astronomical doses of vitamins. It is impossible to induce infantile cortical hyperostosis during the first few months by administering large doses of vitamin A, and affected infants do not have high blood levels of vitamin A.[13]

On the other hand, the disorder known as idiopathic hypercalcaemia is not only considered to follow on excessive vitamin-D administration, but may also be a consequence of abnormal sensitivity to normal intakes of vitamin D or provitamin D.[14]

It was decided to evaluate the rôle of vitamin A in our patient on the basis of the above supposition. Clearly, the disorder is a legacy of the womb, and about the only widespread change in prenatal care during the past 15 years has been the extensive use of (? necessary) vitamins for perfectly well-fed pregnant women.

It was theorized that infants might be sensitized to extraneous vitamin A during foetal life and that, when exposed to vitamin supplements after birth, the disease would become manifest. Our patient's illness began promptly after getting a vitamin preparation.

When the diagnosis was made, the vitamin supplements were stopped, and some 3 weeks later blood was taken and showed the results indicated earlier. Thereafter the patient was encouraged to eat carrots, and was also given a vitamin-A concentrate equivalent to a daily dose of about 1,500 units; a month later blood was again taken. Far from showing an expected exacerbation, there was an improvement, though clinically the bony swellings were unchanged.

SUMMARY

Infantile cortical hyperostosis is a bony disorder of unknown aetiology occurring during the first few months of life. It is a 'new' disease, and probably not too uncommon, even though this is the first case to be recorded locally. Our patient showed the classical features of a hyperostosis affecting the mandible, clavicle and tibiae, as well as other bones. The course of the disease is usually benign, the bony swellings disappearing after some months or years. The lesion may be confined to one bone, and histological features may falsely indicate an osteogenic sarcoma — with tragic consequences. There is no evidence to link the disease with hypervitaminosis A or with an abnormal sensitivity to this vitamin. Present conceptions point to a familial or genetic influence as an aetiological factor.

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ACCIDENT SERVICES OF GREAT BRITAIN AND IRELAND

ABSTRACT OF INTERIM REPORT, 1961

An Accident Services Review Committee was established in 1959 in Great Britain, working in liaison with the sub-committee of the Standing Medical Advisory Committee of the Central Health Services. This review committee has recently published its Interim Report.

The report, a small booklet of 45 pages,[1] is divided into sections. One section deals with the organization of a comprehensive accident service. It stresses the importance of a 24-hour service as its first requirement, and all that this implies, viz. the availability of experienced surgeons, anaesthetists, resident medical officers, nursing, theatre, and laboratory staff, etc. It recommends that the country be zoned into self-contained comprehensive services or 'accident service areas'. Each area is supposed to cover a population of about 1½ million people.

Within each 'accident service area' a three-tier scheme is to operate. The following are the three tiers:

1. The focal point is a Central Accident Unit which should be specially equipped to deal with the intricate treatment of special injuries and it should provide consultant opinion and services for accident problems occurring anywhere within the area.

2. Several Accident Units which should be fully equipped centres for the treatment of major and minor injuries generally.

3. A Peripheral Casualty Service in various forms providing for the treatment of minor injuries arising in the vicinity.

The importance of properly trained ambulance services and ambulance teams to convey patients from the peripheral casualty service to the accident unit or to the central accident unit, is stressed.

The Committee considers the possible merits of an Accident Hospital (a special hospital solely for the treatment of accidental injuries), but is unanimously of the opinion that all accident units of whatever size should be closely associated with general hospitals and should preferably be an integral part of them.

The Peripheral Casualty Service

The purpose of this service is to provide treatment for minor injuries, although it will in addition serve the needs of local casual patients. The siting and staffing of these centres will depend largely on the nature of the area. In general, the service at a cottage hospital or a diagnostic and treatment centre will be staffed by a rota of general practitioners in the area, assisted by nurses, trained ambulance personnel, and voluntary workers. In remote areas without these facilities, minor trauma would have to be treated in the doctor's surgery.

The local general practitioner will be as much a part of the accident service as the staff from the central unit, and this is stressed throughout the report. Now that accidents are a major hazard of modern life, all able-bodied and intelligent persons, particularly in the rural and semi-urban localities, should become acquainted with the principles of first-aid and, as an exercise in citizenship, be enrolled voluntarily on a list of those willing to give their services when called upon. The general practitioner would be the key person in this team.

The Accident Unit

The Committee recommends that an accident unit should always be part of a general hospital, and the unit will be staffed by the general surgeons, orthopaedic surgeons and other specialists on the staff of that hospital, and will be equipped to deal with patients with major injuries, including head injuries, burns, and thoracic, abdominal and vascular injuries. It will work in close cooperation with the central accident unit. The recommendation continues that the ratio of beds in the accident unit should be 25 per 100,000 population, but that the number of units in the accident service area would vary according to the nature of the locality. It will be uneconomical for the unit to have less than 25 beds and undesirable for it to exceed 100 beds.

The bed-occupancy of such an accident unit should be 75% rather than the more usual level of 90%. This will ensure that patients would always be admitted at once to the accident unit without altering the arrangements of the main hospital.

The accident service should communicate directly with the main hospital so that all other medical services that may be required will be available immediately.

Children need special consideration. Whenever possible, they should be admitted to paediatric departments of general hospitals having accident units, to children's hospitals in which there are accident units, or to separate accommodation for children in central accident units or accident units.

The Central Accident Unit

The Committee recommends that there should be one central accident unit, generally serving an area of at least 1,000,000 population, and probably in most cases a maximum of 2 million. This unit will usually be attached to the teaching hospital, although there may be areas where no teaching hospital exists, but in which it will be desirable to establish a central unit. This unit will have 4 functions:

1. To provide for the treatment of patients with multiple injuries who have been transferred from the accident units or elsewhere.

2. To act as a coordinating body for accident arrangements for the region.

3. To provide all services for major and minor injuries to the locality.

4. To undertake undergraduate and postgraduate teaching and research.

Since it must be accepted that this central accident unit will have its being within a teaching hospital, it is expected that the specialists in all the various specialities should be available from the teaching hospital for work within the central accident unit, which naturally will have to work on a 24-hour basis. The closest contact must of course be maintained between the central accident unit and specialist departments in the area.

As regards teaching facilities and teaching requirements in the casualty department, it is stressed that, as far as undergraduate teaching is concerned, a period of at least 3 months should be given which would replace the usual 'few weeks in the casualty department'. As far as postgraduate teaching is concerned it is recommended that the training of all surgeons, regardless of their ultimate specialization, should include a period of training in several of the major accident surgical specialities. At least 6 months of this training should be in an accident unit. 'It is hoped that the Royal Colleges and Corporations will review the requirements for the Diploma of Fellows so as to ensure that every surgeon spends adequate time in the study of trauma before obtaining the Fellowship.'

Finally, the Committee discussed whether it should recommend that accident surgery should be regarded as a speciality with consultants known as accident surgeons. It considers, however, that all surgeons should be trained in the treatment of all the common injuries as part of their basic training for any of the major surgical specialities, including 'general' surgery. The Committee has therefore decided against advising the appointment generally of accident surgeons, and believes that the recommended course of training, which affects the whole of surgery, is a sounder plan. The report concludes with a series of statistical tables setting out the well-known and steady increase of accident death rates year by year. Thus, from 1954 to 1958 the death rate per 100,000 population from motor accidents in males aged 15 - 22 rose from 26 to 35. In addition, an analysis of the most common causes of death showed that accidents of all kinds in the same age period caused 92 deaths per 10,000, whereas cancer caused 114, and cardiovascular disease 142. In the total population up to 85 years of age, accidents caused 174 deaths; this was exceeded only by bronchitis (275), cancer (435), and cardiovascular disease (782).

An appendix gives the floor plans of 4 of the major existing departments in Great Britain, viz. at Sheffield Royal Infirmary; at the Radcliffe Infirmary, Oxford; at the Sunderland Royal Infirmary; and at the Birmingham Accident Hospital.

1. Accident Services Review Committee (1961): *Accident Services of Great Britain and Ireland, Interim Report.* London: B.M.A.

SOUTH AFRICAN MEDICAL AND DENTAL COUNCIL

CANDIDATES FOR ELECTION

The Returning Officer (Mr. W. H. Barnard) has issued the following statement under the date 6 October 1961:

'It is notified in terms of Regulation 3(3) of the First Schedule to the Medical, Dental and Pharmacy Act of 1928 (Act 13 of 1928) as amended, that the following persons have been validly nominated as candidates for election as a member of the South African Medical and Dental Council for the unexpired portion of the quinquennial period ending on 31 December 1963, vice Dr. J. Black, resigned:

Isidore Frack, Johannesburg.
Louis Franklin Freed, Johannesburg.
Jonathan Johan du Pré le Roux, Rosebank, CP.
Tobias Schneider, Johannesburg.
Edwin Wilberforce Turton, Boksburg North.

'As the number of persons so nominated by medical practitioners exceeds the number of persons to be elected, Wednesday 22 November 1961 is appointed by me as being the day on or before which every person entitled to vote at the election as a medical practitioner may sign and transmit or deliver to me a voting paper described in the Third Annexure to the First Schedule of the said Act. A voting paper will be posted to the last registered address of each person qualified to vote at the election.'

Candidates

Invitations to forward information of a personal and professional nature have been extended to all five candidates. The following particulars concerning them have been received:

ISIDORE FRACK, Johannesburg

Dr. Frack was born in Prince Albert, C.P. and brought to Krugersdorp as a young child. He matriculated at Krugersdorp High School and then studied at the University of Pretoria (then T.U.C.). He spent some time as a primary school teacher, and subsequently enrolled at the newly-established medical school of the University of the Witwatersrand. After qualification he practised for 8 years in a Transvaal country town, which later served as the 'Helfontein' of his book, *A South African Doctor looks Backwards and Forwards*. The difficulties, trials, and tribulations of a country doctor are fully recounted, while the question of Poor Whitism is dealt with from a medical and sociological point of view.

After an extensive survey of the conditions in this and surrounding areas, he published a clinical study of chronic goitre.

After relinquishing practice, Dr. Frack attended various clinics and hospitals overseas and resumed private practice in Krugersdorp, where he also became Clinic Medical Officer. On the outbreak of war, he served for close on 7 years as Medical Officer of Health of Krugersdorp. In 1946 he entered the service of the State Health Department and in 1948 was appointed Medical Superintendent of Krugersdorp Hospital. He remained in this post until 1954 when he was appointed Inspector of Hospitals. At various times he was Superintendent in Vereeniging, Coronation, and Boksburg-Benoni Hospitals. He was appointed Superintendent of Baragwanath Hospital in 1957.

LOUIS FRANKLIN FREED, Johannesburg

Degrees. Hons. B.A., M.A. (Psychology, with distinction) (S.A.), D.Phil (Sociology) (Pret.), D.Phil. (Social Science) (O.F.S.), M.B., Ch.B. (St. And.), M.D., D.P.M., D.P.H., D.T.M. & H., D.I.H. (Rand), F.R.S.S.Af., F.R.A.I., F.S.S., F.R.G.S.

Professional and academic appointments. Part-time lecturer on medical sociology and part-time lecturer in the department of psychiatry, University of the Witwatersrand; the first scholar to be appointed an Honorary Visiting Lecturer in the Faculty of Social Science of the University of the Orange Free State; a Fellow of the Royal Society of South Africa; and a Member of the New York Academy of Sciences. He was invited to serve as South African Editor of *Vita Humana: International Journal for Human Development*, Switzerland; was South African Editor of *International Journal of Sexology;* and was a member of the Council of the Southern Transvaal Branch of the Medical Association of South Africa, etc.

Publications. His publications include the following:

1. The Problem of European prostitution in Johannesburg,
2. Sex education in Transvaal schools (1937),
3. The social aspect of venereal disease (1951),
4. The philosophy of sociological medicine (1948),
5. 'Crime', chapter in 'Social medicine', Ed. by Cluver (1951), etc.

JONATHAN JOHAN DU PRÉ LE ROUX, Rosebank, C.P.

Dr. le Roux was born at Ceres, Cape Province, on 3 August 1900. He was educated at the Boys' High School, Paarl, and studied first at the University of Cape Town and then at the University of Edinburgh, where he qualified in medicine in 1923. During 1923 and 1924 he was in private practice in England. He returned to South Africa in 1924 and after further practice at Hermanus, Cape, and elsewhere, he joined the Government service in 1927 as medical officer at the Westfort Leprosy Institution in Pretoria, of which, after obtaining the D.P.H. at the University of the Witwatersrand, he became Medical Superintendent in 1933.

Dr. le Roux left the Government service in 1936, but rejoined in 1939. On 1 March 1952 he was appointed Secretary for Health and Chief Health Officer, from which position he retired on 3 August 1960 on attaining pensionable age.

At the time of his retirement, the President of the South African Medical and Dental Council gave eloquent expression to that Council's appreciation of the excellent and fruitful relations that have been maintained with successive Ministers of Health through Dr. le Roux's mediation. 'Dr. le Roux', said Professor Oosthuizen, 'had been a tower of strength'. Under Dr. le Roux's administration the Medical Association has been able to cooperate harmoniously with the Department of Health and the Ministers in national health affairs.

TOBIAS SCHNEIDER, Johannesburg

Dr. Schneider qualified in 1930 and practised as a general practitioner from 1930 to 1945. Since 1945 he has been in practice in Johannesburg as a specialist physician. He was awarded the Bronze Medal of the Medical Association of South Africa, for meritorious services, in 1960.

Degrees. M.B., B.Ch. (Rand) 1927, M.R.C.P. (Edin.) 1940, M.D. (Rand), F.R.C.P. (Edin.) 1958.

Posts held at present. Physician and Physician-in-charge, Diabetic Clinic, Johannesburg General Hospital, and University of the Witwatersrand; Chairman, Ethical Committee of Federal Council (M.A.S.A.); Member of Federal Council (M.A.S.A.), 1946 to the present; President, Association of Physicians of South Africa; Chairman, Society for Endocrinology, Metabolism and Diabetes of South Africa; Chairman, Press Liaison Committee, Southern Transvaal Branch (M.A.S.A.); Member of Branch Council, Southern Transvaal Branch (M.A.S.A.); Member of Medical Committee, College of Physicians, Surgeons and Gynaecologists of South Africa.

Positions previously held. President, Southern Transvaal Branch (M.A.S.A.); Chairman, Southern Transvaal Branch,

Association of Physicians of South Africa: Hon. Secretary, Southern Transvaal Branch (M.A.S.A.); President, Medical Graduates Association, University of the Witwatersrand; Member of Steering Committee, College of Physicians, Surgeons and Gynaecologists of South Africa: Captain, 25th Field Ambulance, S.A.M.C.; Chairman, General Practitioners War Fund.

EDWIN WILBERFORCE TURTON, Boksburg North

Dr. Turton was educated at Grey College in Bloemfontein where he had a distinguished scholastic and athletic career. He spent his first three academic years at the University of the Witwatersrand and the last three at the University of Cape Town, graduating with the degree of M.B., Ch.B. in 1939.

In 1940 Dr. Turton settled in Boksburg and was appointed part-time general practitioner surgical registrar at the Boksburg-Benoni Hospital until 1945, when he was appointed part-time general practitioner-surgeon at this same hospital. He was a member of the Boksburg-Benoni Hospital Board from 1944 to 1959, and served as Chairman of the Board from 1953 to 1959. His service was terminated in that year by the introduction of the Hospitals Ordinance of 1959 of the Transvaal Province. In 1958 he was appointed a member of the Planning Committee of the Transvaal Provincial Hospital Department.

He has been a member of Federal Council since 1947 to the present day, with a short break from 1952 to 1954. In 1951 he was elected to the Executive Committee of Federal Council and served in this capacity for the year 1951 - 52 and again from 1954 to the present day. He was elected Vice-Chairman of the Council in 1957 and Chairman of Council in 1960.

UNIVERSITEITSNUUS : UNIVERSITY NEWS

UNIVERSITY OF THE WITWATERSRAND MEDICAL GRADUATES ASSOCIATION

BURSARY FUND

The Council of the Medical Graduates Association of the University of Witwatersrand wishes to bring to the notice of those interested that the Alumni Bursary Fund will be available for the year 1962.

The bursary, which was founded by the alumni of the medical faculty, is intended to assist dependents of medical graduates of the University of the Witwatersrand.

Conditions of the award of the bursary are contained in the calendar of the University and may also be obtained from the Secretary of the Association.

Applications, which should reach the undersigned not later than 31 December 1961, should be addressed to: The Hon. Secretary, Medical Graduates Association, Medical School, Johannesburg.

IN DIE VERBYGAAN : PASSING EVENTS

Dr. I. A. Sloan, formerly of Port Elizabeth, is now in specialist anaesthetic practice with Anaesthesia Service Associates, Toronto, Canada, and has been appointed Anaesthetist to the Cardiac Unit, the Hospital for Sick Children; and clinical teacher in anaesthesia at the University of Toronto, Canada.

* * *

Mr. I. Norwich, surgeon, of Johannesburg, has returned after a visit to Europe and Russia, during which he attended the International Surgical Congress in Dublin.

* * *

South African Society of Anaesthetists, Cape Western Branch. The next meeting will be held on Wednesday 1 November at 8.15 p.m. in the small A-floor Lecture Theatre, Groote Schuur Hospital, Observatory, Cape. The subject will be 'Management of thoracic injuries', and a discussion will be led by Mr. R. Hewitson, Dr. J. Ozinsky, and Mr. W. Roberts. All those who are interested are welcome to attend this meeting and to participate in the discussion.

* * *

The South African National Council on Alcoholism has recently printed a small pamphlet outlining the facilities available in South Africa for the care of alcoholics. This pamphlet should be of considerable help to members of the medical profession, and is available on request from the Secretary, South African National Council on Alcoholism, 305 Namaqua House, 36 Burg Street, Cape Town.

* * *

Suidelik-Afrikaanse Hartvereniging: Tak Stellenbosch. Die volgende vergadering is 'n gesamentlike vergadering met die Tak' Kaapstad, en word gehou in die Groot Voorlesingsaal, Karl Bremer-hospitaal, op 2 November 1961 om 8.00 nm. As sprekers tree op dr. L. Vogelpoel (Paradoxical response of the murmurs to the vaso-active drugs in ventricular septum defect with pulmonary hypertension), and dr. F. P. Retief (Aspekte van antistolmiddel terapie in hart- en bloedvattoestande). Na die bespreking sal verversings bedien word.

The College of Physicians, Surgeons and Gynaecologists of South Africa will hold an Admission Ceremony to new Fellows of the College on Friday 3 November in the Great Hall, University of the Witwatersrand, Johannesburg, at 8.30 p.m. At this ceremony, Prof. I. W. Brebner, Emeritus Professor of the University, is to receive an Honorary Fellowship in Surgery. In addition, Mr. B. L. Bernstein, Chairman of the University Council, is to address the gathering.

All those who are interested in attending this ceremony are invited to contact Dr. Phyllis Knocker, 306 Tafelberg, Esselen Street, Hillbrow, Johannesburg, from whom tickets may be obtained.

* * *

British Medical Journal. As announced in the British Medical Journal of 29 July 1961, the Representative Body of the British Medical Association at its meeting in Sheffield this year decided on an increase in the subscription rates to the Journal. Subscription rates for overseas have been increased from £2.12.6 to £3.3.0 per annum as from 1 January 1962.

* * *

Correction. An error occurred in the section on 'Treatment' in the article 'Observations on brucellosis in general practice' by Dr. H. P. Kirsten, which appeared in the issue of the *Journal* for 14 October 1961 (**35,** 858). The sentence (near the top of the second column on p. 859) which reads: 'For the adult, the total dosage varies between 25 - 40 G., being lower or higher at the opposite ends of the age scale', should read: 'For the adult, the total dosage varies between 25 and 40 G., being lower or higher at the opposite ends of the weight scale'.

* * *

The South African Institute for Medical Research, Johannesburg, Staff Scientific Meeting. The next meeting will be held on Monday 6 November at 5.10 p.m. in the Institute Lecture Theatre. Mr. A. K. Lorimer will speak on 'Microbiological assay of vitamins'.

FARMASEUTIESE NUUS : PHARMACEUTICAL NEWS

NEW FILMS FROM BURROUGHS WELLCOME

Burroughs Wellcome & Co. (S.A.) Ltd. announce that 2 new films have been added to their library, the titles and descriptions of which are as follows:

Fundamental Principles of Immunization. 16 mm. colour, sound; running time: 40 minutes. A presentation of some of the basic principles underlying active and passive immunization of human beings and animals with vaccines and antisera. Methods of producing and testing a wide range of prophylactics are illustrated.

The following main subjects are included: Acquirement of natural and artificial immunity; the phenomenon of primary and secondary stimuli in establishing active immunity; passive immunity; transferred immunity in the newborn child and newborn animals; toxoids and killed bacterial vaccines; living attenuated and killed virus vaccines (1961).

Living with Diabetes. 16 mm., colour, sound; running time: 30 mins. The film outlines in simple terms the incidence and mechanism of diabetes and describes in some detail how the diabetic can live a normal, active life through diet alone, or through diet and the use of insulin.

The technique of self-injection of insulin is then demonstrated, and is followed by an illustration of the routine urine test. A brief description of the manufacture of insulin is given, and the final sequences provide examples of some causes and symptoms of hypoglycaemia and hyperglycaemia.

The films are available on loan, free of charge, to the medical and allied professions. Application should be made to Burroughs Wellcome & Co. (S.A.) Ltd., 130 Main Street, Johannesburg. (Telephone No. 22-7324).

NUWE PREPARATE EN TOESTELLE : NEW PREPARATIONS AND APPLIANCES

TREMARIL (WANDER)

Protea Pan Africa Pharmaceuticals Ltd., announce the introduction of Tremaril, and supply the following information:

Tremaril is a novel chemical compound with parasympathicolytic and histaminolytic properties, which effectively calms tremor and loosens muscular rigidity, and provides a means of exerting a favourable influence on extrapyramidal disorders of movement and muscle tone (tremor and rigidity). On tremor (trembling, coarse tremor) in particular, it has a selective sedative action which is independent of its effect of relaxing muscular rigidity. It likewise produces appreciable improvement in the psychic and autonomic disturbances that often accompany Parkinson's syndrome.

Indications. All forms of Parkinson's syndrome: idiopathic, post-encephalitic, and arteriosclerotic parkinsonism, and parkinsonoid manifestations during treatment with neuroplegics; residual symptoms and sequelae in operated parkinsonian patients; senile tremor.

Presentation. Tremaril is supplied in two forms: tablets and bitabs.

Dosage. ½ tablet 3 - 6 times daily, increasing dosage according to patient's requirements.

Contraindications. Intestinal hypotonia and atony, danger of retention of urine (as in prostatic hypertrophy), tachycardiac disorders, and glaucoma.

Further information may be obtained from Protea Pan Africa Pharmaceuticals Ltd., P.O. Box 4699, Johannesburg.

ANATENSOL ELIXIR

Squibb Laboratories announce the introduction of Anatensol Elixir, a new liquid formulation of Anatensol — the tranquillizer that relieves anxiety and tension without clouding consciousness — and supply the following information:

Anatensol Elixir is Squibb fluphenazine dihydrochloride supplied in a palatable, orange-flavoured elixir for 24-hour control of mild, temporary behaviour problems in children. Anatensol Elixir is also intended for 24-hour management of anxiety and tension states in adults who prefer liquid medication or who may have difficulty in swallowing tablets. Each cc. of Anatensol Elixir provides 0·5 mg. of Squibb fluphenazine dihydrochloride.

A highly effective tranquillizer, Anatensol's action is inherently long-acting. It also offers the advantage of control of symptoms at the lowest dosage of any anti-anxiety agent. Thus in most children, an initial dose as low as 0·25 mg. once a day or 0·5 mg. once a day will control symptoms. In adults, the recommended starting dosage is 1 mg. once a day.

Clinical experience has demonstrated that Anatensol has remarkable effectiveness in the treatment of behaviour problems in children, and in managing adult anxiety and tension states caused by environmental situations or produced as an emotional reaction to physical ailments.

In children or adults, Anatensol at the recommended dosages is well tolerated. It should not, however, be used in the presence of severe depression. Anatensol Elixir is available in a 15 cc. bottle with plastic dropper calibrated at 0·5 cc. and 1·0 cc.

Further information can be obtained from Squibb Laboratories (Pty.) Ltd., P.O. Box 48, Isando, Transvaal.

BOEKBESPREKINGS : BOOK REVIEWS

DISEASE AND INJURY

Disease and Injury. Ed. by Leopold Brahdy, M.D. Pp. xviii + 482. Illustrated. R10.00 net. Philadelphia and Montreal: J. B. Lippincott Company. 1961. Distributed by Pitman Medical Publishing Co. Ltd., London.

The 25 contributors to this book are all authorities in their own fields of medical practice, and each in his own field discusses the question — 'Did a given injury cause, precipitate, or aggravate the disease?'

In these days of compensation and litigation the need for medical witnesses to be really expert in their testimony is of much greater importance than it was some years ago. Much the same applies to the certificates which a doctor is called upon to issue almost daily. The chapters of this book are designed to assist the practitioner in these important aspects of his practice.

The late Dr. Samuel Kahn was responsible for a similar symposium in 1937 which was published under the title of 'Trauma and disease.' There have been others with similar names, but this new book contains the latest advances in this subject and is completely up-to-date. A.H.T.

TABAK

Tobacco. Experimental and Clinical Studies. By P. S. Larson, Ph.D., H. B. Haag, M.D., and H. Silvette, Ph.D. Pp. xii + 932. Illustrated. R16.00. Postage: 27½c. London: Baillière, Tindall and Cox Ltd. 1961.

Daar is etlike honderd miljoene rokers in die wêreld vandag. Tabak het vir meer as 300 jaar stadig in gewildheid toegeneem, is as medisyne gebruik, en staan vandag in die regbank, aangeklaag as 'n karsinogeen en kardiovaskulêre gif. As die mediese literatuur oor die laaste dekade bekyk word op hierdie gebied alleen, met die doel om 'n stelling te probeer maak, word jy dikwels gevang. W. B. Bean het in geen onsekere terme, so 'n poging veroordeel as ‚befogged gyrations in the smoke-filled room of his mental processes' (p. 785). Dit geld ook vir sòveel ander studies op die gebied van tabak, dat die skrywers van hierdie monogram die enigste logiese uitweg gekies het: Vertel die hele storie en laat die gevolgtrekkings vir jou leser. Hulle slaag uitmuntend hierin. Uit 1,200 tydskrifte is meer as 6,000 artikels hersien, heeltemal ongeselekteerd. Ook word die polemiek uit briewerubrieke in die wetenskaplike- en leke-pers bygehaal.

Die absorpsie van tabak word bespreek, soos gevind in studies van verskillende organe by verskillende diersoorte en mense. Die effek op orgaansisteme word elk in sy eie hoofstuk bespreek en sluit studies in wat deur die dierewêreld strek vanaf *Bacillus anthracis* tot by *Homo sapiens.*

Hierna word die mens as geheel, sy psige en soma, beskou onder die invloed van tabak.

Die laaste paar hoofstukke word gegroepeer onder die hoof: *Tabak in verhouding tot spesifieke siektes.* Deel I handel meer oor diverse siektetoestande van verskeie orgaansisteme, Deel II bespreek die verhouding tot kardiovaskulêre siektes, en Deel III die verhouding tot die neoplastiese proses.

Verwysings na tegnieke en farmakologies aktiewe bestanddele van tabak is in 2 byvoegsels vervat.

As hierdie bibliografie die les vervat in Bean se woorde: ‚Testimony is not evidence; and evidence is not proof' by lesers van tabak-literatuur tuisbring, het dit reeds baie bereik.

Die boek is 'n naslaanwerk, en hoewel elke geneesheer ongeag sy spesifieke gebied van belangstelling, op een of ander tyd hiervan gebruik sal wil maak, is dit te betwyfel of veel individue hom sal aanskaf. 'n Geneeskundige biblioteek sonder hierdie boek is egter nie toegerus vir naslaanwerk oor tabak — ‚that virtuous herb if opportunely taken.' H.P.W.

FOETAL ELECTROCARDIOGRAPHY

Foetal Electrocardiography. The Electrical Activity of the Foetal Heart. By Saul David Larks, B.S.E.E., M.S. (E.E.), Ph.D. Pp. viii+109. Illustrated. R5.20 Oxford: Blackwell Scientific Publications. 1961.

This newer application of electrocardiography to the study of the foetus will no doubt prove to be a most important advance.

In this book we find the description of the underlying principles of the electromotive forces generated by the foetal heart and also an outline of the technique employed in taking a foetal electrocardiogram. Thereafter the author presents his findings in various stages of normal pregnancy, and outlines experience with abnormal conditions as well as giving a description of the foetal electrocardiogram in animals.

The various aspects discussed are well illustrated with graphs.

Most important appears to be the value of this technique in detecting the presence of foetal life from an early stage, even as early as 11 weeks, also the possibility of defining the presentation of the foetus, the presence of multiple pregnancies, and the behaviour of the foetal heart during actual delivery and under varying conditions of stress.

It appears too that there are definite influences from the maternal side on the behaviour of a foetal heart.

It seems that foetal electrocardiography will offer much to the further understanding of early foetal life, and that it has much to offer obstetricians, physicians, anaesthetists, and cardiologists alike. A.J.B.

PATHOLOGY OF IONIZING RADIATION

The Pathology of Ionizing Radiation. By Shields Warren, M.D., Sc.D., LL.D. Pp. ix+42. Illustrated. R2.40 net. Oxford: Blackwell Scientific Publications. 1961.

The author is a distinguished pathologist who has been closely associated with the medical aspects of the development of the atomic bomb. The monograph based on lectures the author has given on the subject, is very concise. It consists, with the references, of 40 pages. There is, however, a great deal of information packed into this small volume on the subject of the acute radiation syndrome and the pathological effects of ionizing radiation. In the present atomic age, every senior medical student and every doctor must have some knowledge of the subject of the effects of ionizing radiation. The book is easily read and can be read through in a matter of two hours. Every medical student, after he has completed his course in pathology, should be able to follow the description of the pathological effect of ionizing radiation, and it follows that every senior medical student and general practitioner and specialist should read this monograph. Every diagnostic radiologist should have this monograph, and even for the radiotherapy specialist it forms a very good summary. The monograph is excellently prepared and it is a pleasure to read it. M.W.

BRIEWERUBRIEK : CORRESPONDENCE

RECIPROCAL EXCHANGE OF TEACHERS AND RESEARCH WORKERS

To the Editor: The recent Royal Commission, which enquired into the working of the National Health Service in Britain, recommended that registrars and junior consultants might accept appointments abroad for 2 or 3 years without fear of losing their seniority in the Service. This means that it is now possible to arrange for an exchange of teachers and research workers between the older medical schools of Britain and the younger ones in the less-developed countries. It may well be that this two-way exchange of experience may become in the future an essential qualification for the higher teaching appointments both in Britain and abroad.

The Medical Associations of Canada and South Africa have attained independence of their parent Association, and Australia and New Zealand may follow their example next year. Nevertheless, they will remain affiliated members of their parent Association.

It is true that the World Medical Association acts as a forum for the exchange of international ideas, but this is no substitute for an affiliation that aims at the preservation of the best that British Medicine can offer by means of a reciprocal exchange of teachers and research workers. It is therefore imperative that South African medicine should be strongly represented next year at the Congress in Colombo in order that our Association may continue to enjoy the advantages of affiliation with the older medical schools and research centres in Britain.

Sanlam Buildings B. Crowhurst Archer
Durban
11 October 1961

LIMITS FOR GENERAL PRACTITIONERS

To the Editor: We were glad that Dr. Hamilton chose to express himself so wisely and vividly in the *Journal* of 30 September 1961.[1] Of course such limits must be related to the needs and circumstances of people and to the places in which we practise. Whatever the heights which medicine and surgery may reach in the leading centres in South Africa, the fact remains that there are millions of its inhabitants suffering the basic hazards of life and health for whom there is very little hospital accommodation and very few medical staff. When, in the course of time, this striking imbalance is corrected, we suppose we may no longer be bound to give anaesthetics, operate for obstructed labours, relieve the acute abdomen, deal with the complications of prolonged middle-ear infection, evacuate the extradural haemorrhages, and so forth.

Dr. Hamilton refers to the advances in medicine as well as surgery. Current interest dwells on the limits and excesses of the ordinary surgeon, though much might as well be said about the uses and abuses of medical therapy. Who is sufficiently experienced to diagnose valvular disease of the heart accurately, and then prescribe digitalis and the modern diuretics properly? Who is in a proper position to use the modern synthetic hormones?

We agree completely that 'the correct approach to the problem is to train all doctors into a realization of their own capabilities and limitations' and that 'the Council is already fully equipped to deal with transgressions'. Surely the Council's collective opinion is wise enough to bear these realities in mind?

We recall the inscription on the flyleaf of Hamilton Bailey's *Emergency Surgery:* 'A true surgeon is never fearless. He fears for his patients, he fears for his shortcomings, his own mistakes, but he never fears for himself or his professional reputation.' This is indeed the real sense in which those who, of necessity, continue to be average general surgeons should circumscribe their practice.

R. F. Ingle
P. C. Ingle

All Saints Mission Hospital
P.O. All Saints
Transkei
7 October 1961

1. Correspondence (1961): S. Afr. Med. J., 35, 824.